

Workplace Exposure to Passive Smoking and Risk of Cardiovascular Disease: Summary of Epidemiologic Studies

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We reviewed the published epidemiologic studies addressing the relationship between workplace exposure to environmental tobacco smoke (ETS) and cardiovascular disease risk in three case-control studies and three cohort studies. Although the point estimates of risk for cardiovascular disease exceeded 1.0 in five of six studies, none of the relative risks was statistically significant because of the small number of cardiovascular end points occurring in individual studies. In common with most epidemiologic investigations of the health risks of ETS, none of the workplace studies included independent biochemical validation of ETS exposure. In contrast to the evidence on increased cardiovascular disease risk from exposure to spousal ETS, studies of ETS exposure in the workplace are still sparse and inconclusive. Conversely, there is no biologically plausible reason to believe that the hazards of ETS exposure that have been demonstrated in the home should not also apply to the workplace. *Key words:* cardiovascular disease, environmental tobacco smoke, passive smoking, tobacco smoke pollution. — *Environ Health Perspect* 107(suppl 6):847–851 (1999).

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Epidemiologic Studies of Workplace Exposure to Environmental Tobacco Smoke and Cardiovascular Disease

This article reviews the methods and findings of published studies addressing the relationship between workplace exposure to environmental tobacco smoke (ETS) and the risk of cardiovascular disease. Using the MEDLINE database (National Library of Medicine, Bethesda, MD), we conducted a search of the published peer-reviewed literature linking ETS to cardiovascular disease. All studies located in this database were reviewed to determine if the investigators specifically examined workplace exposure to ETS. We identified six published epidemiologic studies—three case-control studies (1–3) and three cohort studies (4–6)—that examined the relationship between ETS exposure in the workplace and risk of cardiovascular disease.

Considering current exposure to ETS in the workplace, the point estimates of the odds ratios/relative risks for cardiovascular disease exceeded 1.0 (range, 1.2–1.9) in five of the six studies, although none of the estimates was statistically significant (Table 1, Figure 1). The imprecision of the risk estimates (with the exception of the large American Cancer Society CPS-II cohort) (5) reflects the relatively small number of cardiovascular events occurring in the individual studies.

Recently, Wells (7) reported a meta-analysis of the published and unpublished studies on the relationship of passive smoking in the workplace and risk of cardiovascu-

lar disease. In addition to the six published studies included in this survey (1–6), the meta-analysis by Wells included data from two unpublished doctoral dissertations (8,9). These eight studies yielded a pooled relative risk estimate of 1.18 (95% confidence interval [CI], 1.04–1.34) (7). Since the purpose of the present review was not to replicate Wells' meta-analysis, we will not further consider the unpublished dissertations. We will review the six published studies (1–6) from the perspective of their adequacy in measuring ETS exposure at work, addressing issues of confounding, and assessing dose-response relations.

Assessment of ETS Exposure

Environmental tobacco smoke exposure was assessed in all studies by interview (case-control studies) or self-completed questionnaire (cohort studies). With the exception of the case-control study by Dobson et al. (1), none of the studies included biochemical validation of ETS exposure. Dobson et al. (1) measured plasma fibrinogen levels while ascertaining ETS exposure. They reported higher fibrinogen levels among individuals reporting ETS exposure than among those reporting no exposure, although the differences were not statistically significant. Investigators in only two of the six studies (2,6) sought and reported information regarding the intensity of exposure to ETS at work (Table 2). Both studies reported a dose-response relation between higher intensity of ETS exposure in the workplace and risk of cardiovascular disease. In only two of the six studies (2,3), information was collected and analyzed on the duration of exposure to ETS in the workplace, and neither study found an association

between increasing duration of exposure and risk of cardiovascular disease.

Several types of exposure misclassification could have potentially occurred in the six cited studies (10):

- Misclassification of smokers falsely reporting themselves to be nonsmokers. This type of misclassification is believed to be minor. For example, the proportion of individuals in observational studies who falsely report not smoking but who are assessed to be smokers by cotinine or nicotine measurement in body fluids ranges from 1–4% (11).
- Under- or overestimation of ETS exposure among nonsmokers. The extent to which recall bias might result in overestimation of ETS exposure in case-control studies is unknown. Generally, nonsmokers are more likely to underestimate their extent of ETS exposure. For example, in a study of 663 never smokers and former smokers attending a cancer screening clinic, 91% had detectable levels of cotinine in their urine even though only 76% of subjects reported ETS exposure in the previous 4 days (12). Although there are dietary and other noninhaled sources of nicotine, these are likely to make a negligible contribution to cotinine levels (13,14).
- Inaccuracy of self-reported intensity of exposure to ETS. Self-reported current exposure to passive smoking correlates only modestly (Pearson coefficients ranging between 0.2 and 0.5) with biochemical markers such as salivary (12) and urinary (15) cotinine. This is likely to result in a bias toward the null in estimates of dose-response relations between intensity of ETS exposure and risk of disease.
- Downward secular trends in ETS exposure. The prevalence of passive smoking has declined during the past decade in countries such as the United States since

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Table 1. Summary of studies examining current exposure to ETS in the workplace in relation to cardiovascular disease risk.

Study (reference)	No. of cases	Crude OR/RR (95% CI)	Adjusted OR/RR (95% CI)	Confounders adjusted for
Case-control studies^a				
Dobson et al., 1991 (1)	M 27 F 5	0.90 (0.50–1.60) 0.71 (0.19–2.27)	0.95 (0.51–1.78) 0.66 (0.17–2.62)	Age, past history of coronary heart disease
He et al., 1994 (2)	F 33	2.45 (1.23–4.88)	1.85 (0.86–4.10)	Age, hypertension, type A, serum total, cholesterol, high-density lipoprotein, ETS from spouse
Muscat and Wynder, 1995 (3)	M 44 F 20	Not stated Not stated	1.2 (0.6–2.2) 1.0 (0.4–2.5)	Age, education, hypertension
Cohort studies^b				
Svensden et al., 1987 (4)	M 69	1.4 (0.80–2.50)	Not stated	Age, wife's smoking status
Steenland et al., 1996 (5)	M 768 F 319	Not stated Not stated	1.03 (0.89–1.19) 1.06 (0.84–1.34)	Hypertension, diabetes, BMI, education, aspirin use, alcohol, exercise, employment status
Kawachi et al., 1997 (6)	F 64	1.53 ^c (0.73–3.18) 2.16 ^d (1.02–4.58)	1.49 (0.71–3.14) 1.92 (0.88–4.18)	Age, hypertension, diabetes, hypercholesterolemia, BMI, aspirin use, alcohol, vitamin E, BMI, PMH use, menopausal status, past oral contraceptive use, saturated fat intake, family history of myocardial infarction, father's occupation

Abbreviations: BMI, body mass index; F, female; M, male; OR, odds ratio; PMH, postmenopausal hormone; RR, relative risk. ^aExposed cases. ^bCardiovascular cases. ^cOccasional ETS exposure. ^dRegular ETS exposure.

increasing numbers of people have stopped smoking and more workplaces have become smoke free. Cohort studies that assessed ETS exposure in the 1970s and mid-1980s, and only once at baseline (4–6) (Table 3), would continue to count individuals as exposed even though exposure ceased during the course of the study and these individuals presumably benefited from reduced risks of cardiovascular disease. In other words, these studies probably have underestimated the risk of disease.

Confounding

Individuals exposed to passive smoking at home are less healthy with respect to other lifestyle habits compared to unexposed individuals (10,16,17). However, little has been documented about differences in health habits comparing those individuals exposed to ETS in the workplace to those not exposed. It is likely, however, that voluntary restrictions on workplace smoking are less common in blue-collar occupations and that workers employed in these jobs tend to have fewer health-conscious health habits than white-collar workers.

Given the relatively modest associations between passive smoking and cardiovascular disease, it is important to exclude confounding as an explanation for the observed association. Of the studies that examined ETS exposure in the workplace and cardiovascular disease, only three (2,5,6) managed to adjust for a reasonably broad range of potential

confounding factors (Table 1). In all three studies, adjustment for confounding factors resulted in some reduction of the risk estimates. In the case-control study by He et al. (2), adjustment for multiple risk factors for cardiovascular disease (including history of hypertension, type A personality, total and high-density lipoprotein [HDL] cholesterol levels, and passive smoking at home) resulted in a 24% reduction in the odds ratio for cardiovascular disease. However, to the extent that a reduction in HDL cholesterol may lie in the causal pathway between passive smoking and cardiovascular disease (18), adjusting for this variable may have resulted in statistical overcontrol. However, because only the final adjusted model was presented in the study by He et al. (2), it was not possible to assess the extent of this problem. In two cohort studies (5,6), adjustment for confounding resulted in a very modest reduction (2–11%) in the relative risks (Table 1). Overall, unadjusted confounding is believed to result in a small bias in overestimating the risk of passive smoking on cardiovascular disease (10,18,19).

Ascertainment of Disease End Points

In total, the evidence on ETS exposure in the workplace and risk of cardiovascular disease is based upon 1,594 end points, to which the American Cancer Society CPS-II cohort (5) contributed 68% of cases (Table 3). The six studies used different definitions of

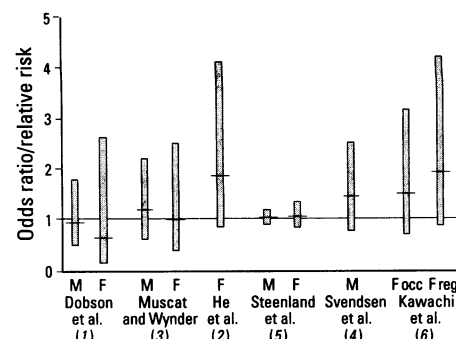


Figure 1. Studies of ETS exposure in the workplace and cardiovascular disease. Point estimates of odds ratios/relative risks and 95% confidence intervals. Abbreviations: F, female; M, male; occ, occasional exposure to workplace ETS; reg, regular exposure to workplace ETS.

cardiovascular disease end points. Three studies used World Health Organization (WHO) criteria (i.e., symptoms plus either cardiac enzyme level elevations or diagnostic electrocardiogram changes) (20) to confirm cardiovascular disease end points (1,2,6); two studies (3,5) used the *International Classification of Diseases, 9th revision* codes (21); in one case, the Multiple Risk Factor Intervention Factor study (4) used internal criteria developed by a panel of cardiologists and physicians. Four studies (2–4,6) restricted their end points to incident events occurring among subjects free of a history of heart disease; two studies (1,5) included disease events occurring among those with a history of heart disease. Forty-two percent of the end points in the study by He et al. (2) comprised cases of myocardial infarction confirmed by WHO criteria; the remainder (58%) comprised cases of coronary stenosis (> 50% occlusion) diagnosed by angiography. It is possible that such variations in the definition of end points may have led to discrepancies in the association of passive smoking and coronary disease. Despite the heterogeneity in case definition, however, we discerned no obvious pattern of differences in results according to the end point adopted. None of the six studies reported relationships of ETS exposure in the workplace to other cardiovascular disease end points such as stroke or peripheral vascular disease.

Dose-Response Relations between ETS Exposure and Risk of Cardiovascular Disease

Intensity of ETS Exposure in the Workplace

Only two studies collected information on intensity of ETS exposure in the workplace. He et al. (2) assessed the number of smoking co-workers, the estimated number of cigarettes

smoked per day, and the average duration of exposure (number of hours per day). Kawachi et al. (6) asked respondents to self-rate their exposure at work as "never," "occasional," or "regular." Steenland et al. (5) also asked about the number of hours per day of ETS exposure in the workplace but did not report their findings. Both the case-control study by He et al. (2) and the study by Kawachi et al. (based on the Nurses' Health study) (6) found evidence of dose-response relations between intensity of ETS exposure in the workplace and risk of cardiovascular disease (Table 4).

Duration of ETS Exposure in the Workplace

Duration of ETS exposure in the workplace was assessed in only two studies. He et al. (2) requested number of years of exposure at work; Muscat and Wynder (3) requested number of years of exposure to ETS as an adult but did not separate home exposure from work exposure. In the case-control study by He et al. (2), none of the women had been exposed for less than 5 years. Both the American Cancer Society CPS-II cohort (5) and the Nurses' Health study (6) asked about duration of exposure to spousal smoking but did not assess duration to workplace ETS exposure. He et al. (2) did not find a statistically significant trend between duration of ETS exposure and risk of cardiovascular disease; Muscat and Wynder (3) concluded that there was no trend between duration and risk of heart disease, although they did not report a formal test for trend (Table 4).

The Discrepancy between Dose-Response Relations Comparing Intensity to Duration

The discrepancy between dose-response trends comparing intensity and duration of ETS exposure may result from greater misclassification of cumulative duration of exposure compared to assessment of current

Table 2. Environmental tobacco smoke exposure assessment in studies of exposure in the workplace and risk of cardiovascular disease.

Study (reference)	Method of assessment	Prevalence of exposure	Comments
Case-control studies^a			
Dobson et al., 1991 (1)	In-hospital interview (nonfatal myocardial infarction)	Home M 13% F 19%	No data on intensity/duration of exposure
	Questionnaires sent to relatives (fatal coronary heart disease)	Work M 44% F 37%	Plasma fibrinogen measured at same time
He et al., 1994 (2)	Standardized interview	Home F 45% Work F 32%	Ascertained intensity (no. of smokers; no. of cigarettes smoked/day by others; no. of hours exposed/day) and duration (no. of years exposed) Intensity showed dose-response relation to disease risk; duration did not No biochemical validation Test-retest interview conducted in 33% of cases; 74% agreement for passive smoking at work
Muscat and Wynder, 1995 (3)	In-hospital interview	Home M not stated F 24% Work M 61% F 44%	No data on intensity Duration ascertained but not analyzed separately from home exposure No biochemical validation
Cohort studies^b			
Svensen et al., 1987 (4)	Baseline questionnaire (1973)	Home M 20% Work M 73%	No updating of exposure during 8-yr follow-up Workplace ETS exposure assessed by asking subjects about smoking status of "most of their co-workers"
Steenland et al., 1996 (5)	Baseline questionnaire (1982)	Home M 19% F 28% Work Not stated	No updating of exposure during 8-yr follow-up Assessed intensity (no. of hours per day), but not reported 28% of men and 48% of women missing data on ETS exposure at work; excluded from analyses
Kawachi et al., 1997 (6)	Baseline questionnaire (1982)	Home F 42% Work F 78% (66% occasional, 33% regular)	No updating of exposure during 10-yr follow-up Assessed intensity but not duration of exposure at work Intensity showed dose-response relation

^aPrevalence of exposure in smokers. ^bPrevalence of exposure in nonsmokers.

Table 3. Cardiovascular disease end points examined in studies of ETS exposure in the workplace.

Study (reference)	End points	Confirmatory criteria	Comments
Dobson et al., 1991 (1)	Nonfatal myocardial infarction Coronary death	WHO criteria	No breakdown of causes (n = 201 total) Included cases among those with history of coronary heart disease
He et al., 1994 (2)	Nonfatal myocardial infarction (n = 25); > 50% coronary stenosis (n = 34)	WHO criteria Angiography	Incident cases
Muscat and Wynder, 1995 (3)	Nonfatal myocardial infarction (n = 114)	ICD-9 code 410.0 ^a	Incident cases
Svensen et al., 1987 (4)	Fatal (n = 13) or nonfatal (n = 56) coronary heart disease	Classified by three cardiologists, based on review of hospital records, autopsy reports, death certificates	Incident cases
Steenland et al., 1996 (5)	Coronary heart disease death (n = 1,087)	Death certificates coded to ICD-9 (codes 410-414) ^a	Included 5-10% cases with history of coronary heart disease
Kawachi et al., 1997 (6)	Nonfatal myocardial infarction (n = 52); fatal coronary heart disease (n = 12)	WHO criteria Death certificates	Incident cases

^aInternational Classification of Diseases, 9th revision (21).

Table 4. Dose-response relations (intensity and duration) between ETS exposure in the workplace and cardiovascular disease risk.

Study (reference)	Exposure category	No. of exposed cases	Adjusted OR/RR	p, trend
Intensity				
He et al., 1994 (2)	No. of cigarettes smoked/day by co-workers			
	0-5 (actually 0)	26	1.00	
	6-10	10	0.87 (0.30-2.53)	
	11-20	15	2.95 (1.05-8.28)	
	20+	8	3.56 (0.81-15.6)	0.02
	No. of smoking co-workers			
	0	26	1.00	
	1-2	16	1.16 (0.48-2.82)	
	3	12	5.06 (1.42-18.0)	
	4+	5	4.11 (0.39-43.7)	0.02
	No. of hours/day			
	0	26	1.00	
Kawachi et al., 1997 (6)	Never	9	1.00	
	Occasional	32	1.49 (0.71-3.14)	
	Regular	23	1.92 (0.88-4.18)	0.04
Duration				
He et al., 1994 (2)	No. of years exposed			
	0-5 (actually 0)	26	1.00	
	6-15	8	3.08 (0.90-10.6)	
	16+	25	1.56 (0.67-3.64)	0.12
Muscat and Wynder, 1995 (3) ^a	No. of years exposed			
	Male			
	0	38	1.0	
	1-20	12	1.7 (0.7-4.5)	
	21-30	5	1.5 (0.4-5.2)	
	31+	13	1.1 (0.4-2.8)	NS
	Female			
	0	13	1.0	
	1-20	12	2.0 (0.5-8.1)	
	21-30	5	0.9 (0.2-4.4)	
	31+	16	1.7 (0.5-5.9)	NS

NS, not stated. ^aCombined exposure to ETS in the home and workplace.

intensity of exposure and/or the possibility that ETS increases cardiovascular disease risk via acute mechanisms so that increasing duration of exposure does not contribute to further increases in risk.

In assessment of current ETS exposure compared to cumulative duration of exposure, Pron et al. (22) found that self-reported duration of exposure was less reliable than the assessment of recent passive smoking exposure. In that study, a correlation coefficient of 0.25 was found between the reported durations of exposure to spousal smoking at the initial and repeat interviews. When misclassification of exposure duration is minimized, it may be possible to observe a dose-response relation to cardiovascular disease risk. For example, Steenland et al. (5) found evidence for a dose-response relation between duration of home exposure to ETS and risk of cardiovascular disease risk when their analysis was restricted to the subgroup of the American Cancer Society CPS-II cohort in whom reports of exposure to ETS were concordant for both self-reports and spousal reports.

Alternatively, some authors have suggested that the mechanisms by which ETS increases the risk of cardiovascular disease are short-term (in which case, increasing duration of ETS exposure may not produce increasing risk of disease). On the basis of data from published experimental studies, Law et al. (23) calculated effects of tobacco smoke exposure on platelet aggregation. In experiments, exposure to second-hand smoke for 20 min resulted in a 1.03-standard deviation increase in platelet aggregation, whereas actually smoking one to two cigarettes was associated with about a 1.25-standard deviation increase (24). The effect of tobacco smoke exposure on platelet aggregation is believed to be acute; 1 hr after a single exposure, the effect on platelet aggregation is nearly halved. Working from this experimental evidence, Law et al. (23) calculated that repeated exposure to tobacco smoke throughout the day (such as experienced by a regular passive smoker) could be consistent with a 20-30% elevation in risk of cardiovascular disease.

Workplace Exposure Contrasted to Home Exposure

For both men and women, the prevalence of exposure to ETS in the workplace was substantially higher than in the home in these six studies (Table 2). The sole exception was the study from China by He et al. (2) in which women reported a higher prevalence of exposure to ETS at home than at work. None of the studies assessed the intensity of ETS exposure at home and in the workplace, so that it is possible that home exposure may be more intense than workplace exposure (25,26). Conversely, environmental monitoring studies suggest that for many groups of workers occupational exposure may involve a higher density of smokers in the immediate environment (27,28). Additionally, many individuals spend more time at work than in the company of their spouses at home.

Despite the higher prevalence of ETS exposure in the workplace than in the home, the evidence linking ETS to cardiovascular disease is more consistent and abundant for home exposure. The reason for the wealth of evidence for home exposure is because many epidemiologic studies have been able to use proxy measures of exposure to spousal tobacco smoke (i.e., using "married to a smoker" as an indicator of ETS exposure at home). The point estimates for home and workplace exposure are similar, however, and the 95% CI values overlap in most cases. For example, in the study by Kawachi et al. (6), among women exposed only at work and not at home, the adjusted relative risks of total coronary heart disease (CHD) were 1.49 (95% CI, 0.71-3.14) among those occasionally exposed and 1.92 (95% CI, 0.88-4.18) among those regularly exposed to ETS. Among women exposed only at home and not at work, the corresponding adjusted relative risks were 1.19 (95% CI, 0.63-2.31) and 2.11 (95% CI, 1.03-4.33). In the case-control study by He et al. (2), the adjusted odds ratio of coronary disease among women exposed to ETS at home was 1.24 (95% CI, 0.56-2.72) compared to 1.85 (95% CI, 0.86-4.00) for exposure in the workplace. He et al. (2) also reported additive effects of home and workplace exposure on risk of coronary disease.

A potential problem in contrasting home and workplace exposure is the handling of simultaneous exposure in both settings. In the study by Kawachi et al. (6), women reporting any ETS exposure at home were excluded from the workplace analyses and vice versa. In the case-control study by He et al. (2), analyses of workplace ETS statistically adjusted for home exposure. However, in the remaining three studies (1,3,5), it is unclear how the investigators handled simultaneous exposure; i.e., there may have been some misclassification

of exposure if a proportion of individuals were exposed in both settings, and this was not taken into account.

Effects of Workplace ETS Exposure in Population Subgroups

Gender

Of the 1,594 end points included in the five studies, 515 (32%) occurred among women. Five studies (1–3,5,6) included women (Table 1); four studies (1,3–5) included men. The data are too sparse to draw any conclusions about gender differences in risk of ETS exposure in the workplace.

Social Class

Steenland et al. (5) conducted separate analyses for blue-collar and white-collar workers and found that passive smoking in the workplace was related to CHD risk only in male blue-collar workers (adjusted relative risk, 1.36; 95% CI, 1.01–1.83). ETS exposure was not related to CHD risk among white-collar workers, male or female.

Former Smokers

Dobson et al. (1) analyzed the risks of ETS exposure in the workplace separately for never smokers and former smokers and found no relationship between passive smoking and risk of CHD among either group for males. Among females, the adjusted odds ratios of CHD from ETS exposure was 0.66 (95% CI, 0.17–2.62) among never smokers and 2.21 (95% CI, 0.33–14.9) among former smokers. However, as the width of the 95% CI values attests, the analyses among women were based on a few exposed cases (five each among never smokers and former smokers) (1).

Conclusions

In contrast to the evidence on spousal exposure to ETS and cardiovascular disease, studies of ETS exposure in the workplace are still sparse and inconclusive. Although the point estimates of risk for cardiovascular disease exceeded 1.0 in five of six studies, none of the relative risks was statistically significant

because of the small number of cardiovascular end points occurring in individual studies (except for the American Cancer Society CPS-II study). All the published studies assessed ETS exposure in the workplace by self-report (interviews or questionnaires). In common with most epidemiologic studies of ETS, none of the workplace studies provided independent biochemical validation of ETS exposure. However, most exposure misclassifications in these studies are likely to be minor and may have introduced a conservative bias in the estimation of risk. Potential confounding is possibly a more important issue in studies of cardiovascular disease than in studies of lung cancer, but three of the six published studies adjusted for a broad range of potential confounding factors, and two of these found evidence of increased risk of cardiovascular disease with ETS exposure in the workplace. Further studies are needed to conclusively demonstrate an effect of ETS exposure in the workplace on cardiovascular disease risk. In particular, studies are needed to relate the intensity and duration of ETS exposure at work to cardiovascular disease risk. Despite the sparsity of evidence on workplace exposure, however, there is little biologically plausible reason to believe that the hazards of ETS exposure that have been demonstrated in the home setting should not also apply to the work setting.

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